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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/640,952	08/17/2000	Michael S. Kinch	3220-66872	3252

26813 7590 04/21/2004

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EXAMINER	
CANELLA, KAREN A	

ART UNIT	PAPER NUMBER
1642	

DATE MAILED: 04/21/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/640,952	KINCH ET AL.	
	Examiner	Art Unit	
	Karen A Canella	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on _____.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☐ Claim(s) *1,3-13,21,23,24,28,30,33-37,50-69,72,73,75-81,90 and 91* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☐ Claim(s) *1,3-7,9-12,21,23,24,28,30,33-37,50-58,61,64,66-69,72,73,75-81,90 and 91* is/are rejected.

7)☐ Claim(s) *8,13,36,37,42,59,60,62,63 and 65* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *5/02+12/03+10/01*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Claims 1, 3, 5, 21, 33, 34, 35, 36, 37, , 41, 43, 45, 46, and 91 have been amended. Claims 31 and 38-49 have been canceled. Claims 1, 3-13, 21, 23, 24, 28, 30, 33-37, 50-69, 72, 73, 75-81, 90 and 91 are pending and under consideration.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office Action

Claims 4, 50, 54, 77 and 81 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 4, 50, 54, 77 and 81 are rejected as drawn to the laboratory designation of D7 and B2D6 as the sole means of identifying the antibodies on which the instant method claims rely. The use of laboratory designations only to identify a particular antibody/cell line renders the claims indefinite because different laboratories may use the same laboratory designations to define completely distinct hybridomas and antibodies. Amendment of the claims to include the depository accession number of the mAb or hybridoma is required, because deposit accession numbers are unique identifiers which unambiguously define a given hybridoma and/or monoclonal antibody.

It is unclear how claim 24 further limits claim 21. Claim 21 has been amended to read on detecting reagent-nucleic acid binding. Claim 24 recites immunfluorescence staining. It is unclear if claim 21 intends the immunofluorescent stain to bind to the EphA2 protein in an auxiliary method step, or if claim 24 encompasses a step wherein the reagent which is bound to the nucleic acid is detected by immunofluorescent staining. For purpose of examination, both alternatives will be considered.

The metes and bounds of claim 91 are unclear. The art teaches that normal cells do not express, or express very low levels of, Epha2. Claim 90 recites the specific limitation of a change relative to a normal cell population. Claim 91 embodies the method of claim 90 wherein a change is indicative of non-metastatic cancer cells in said population. However, if primary non-metastatic cancer cells were measured they would have levels of EphA2 commensurate with

the normal cells and would not exhibit "a change", so the metes and bounds of claim 91 cannot be discerned.

Claims 4, 50, 54 , 77 and 81 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. Applicant has filed a Declaration regarding the deposit of hybridoma cell line producing the antibody D7 and B2D6 , however, applicants declaration is directed to 09/640,935 and not the instant 09/640,952.

Claims 21, 23, 90 and 91 are rejected under 35 U.S.C. 102(b) as being anticipated by Easty et al (International Journal of Cancer, 1995, vol. 60, pp. 129-136) as evidenced by the abstract of Chen et al (Journal of Biological Chemistry, 1998, Vol. 273, pp. 24670-24675) .

Claim 21 is drawn to a method for detecting the presence of metastatic cells in a tissue sample comprising the steps of incubating the tissue sample with a reagent capable of specific binding to a nucleic acid coding for the EphA2 protein to allow reagent binding to the nucleic acid and detecting reagent-nucleic acid binding, wherein said binding is indicative of the presence of metastatic cancer cells. Claim 23 embodies the method of claim 21 wherein the nucleic acid is DNA or RNA.

Claim 90 is drawn in part to a method for detecting the presence of cancer cells in a selected cell population comprising assaying at least a portion of the selected cell population for a change in EphA2 expression level as compared to EphA2 expression level in an analogous normal cell population, wherein he assaying the cell population comprises incubating at least a portion of the selected cell population with a monoclonal antibody and wherein the change is indicative of the presence of cancer cells in the selected cell population. Claim 91 embodies the method of claim 90 wherein the change in EphA2 expression level is indicative of the presence of non-metastatic cells in the cell population. It is noted that the metes and bounds of claim 91 cannot be discerned for the reason set forth under the rejection under 112 second paragraph above.

The abstract of Chen et al discloses that EphA2 is synonymous with Eck

Easty et al disclose a method for detecting metastatic melanoma cells comprising detecting mRNA for Eck.(Table II and page 134, column 1, line 13 to column 2, line 4) and protein from Eck (page 131, first column, under the heading "Immunoblotting analysis), thus fulfilling the specific embodiments of claims 21, 23 and 90, as drawn to Epha2 expression. Easty et al disclose the detection of Eck by immunoblotting (page 131, column 1, under the heading "Immunoblotting analysis) and immunohistochemistry (page 135, second column, first full paragraph), thus fulfilling the specific embodiments of claim 24.

Claims 1, 3, 5, 11, 33, 34, 35, 41, 45, 47, 49, 55, 56, 57, 58, 61, 64 and 68 are rejected under 35 U.S.C. 103(a) as being unpatentable over Easty et al (International Journal of Cancer, 1995, vol. 60, pp. 129-136) as evidenced by the abstract of Chen et al (Journal of Biological Chemistry, 1998, Vol. 273, pp. 24670-24675) and Lindberg et al (Molecular and Cellular biology, 1990, vol. 10, pp. 6316-6324) in view of Larrick et al (In: Human Hybridomas and Monoclonal Antibodies, Engleman and Foung, Ed.s, 1985, pp. 8-9).

Claim 1 is drawn to a method for detecting the presence of metastatic cells in a cell population comprising the steps of lysing at least a portion of the cell population, incubating the lysed cells with monoclonal antibody that specifically binds to EphA2 to allow antibody binding to EphA2 and detecting antibody-EphA2 binding. Claim 3 embodies the method of claim 1, wherein antibody binds to an intracellular epitope of EphA2. Claim 5 embodies the method of claim 1 wherein the antibody is labeled with a detectable label. Claim 11 embodies the method of claim 1 wherein the incubating and detecting steps comprise Western Blotting methodology. Claim 33 embodies the method of claim 5 wherein the antibody comprises at least one of a fluorescent, chemiluminescent, bioluminescent, enzymatic, chromogenic or radioactive label, and wherein detecting the antibody-EphA2 binding comprises detecting at least one detectable label. Claim 34 embodies the method of claim 1 wherein the cell population comprises cells selected from the group consisting of lung cells and colon cells. Claims 35 embodies the method of claim 1 wherein the cell population comprises epithelial cells. Claim 41 embodies the method of claim 1 wherein the cell population comprise cells from a tissue biopsy.. Claim 45 embodies the method of claim 1 wherein detecting the antibody-EphA2 binding comprises utilizing a

diagnostic method selected from the group consisting of an ELISA assay, a Western blot and flow cytometry.

Claim 47 is drawn to a method for detecting the presence of metastatic cells in a cell population comprising incubating at least a portion of the cell population with a monoclonal antibody that specifically binds EphA2 to allow binding of the antibody to EphA2 and detecting antibody EphA2 binding, wherein antibody AphA2 binding is indicative of the presence of metastatic cells in the cell population. Claim 49 is drawn to the method of claim 47 wherein the antibody bind to an intracellular epitope of EphA2. Claim 55 embodies the method of claim 47 wherein the bound antibody comprises a detectable label, and wherein detecting antibody-EphA2 binding comprises detecting the label. Claim 56 embodies the method of claim m47 wherein the bound antibody comprises at least one of a fluorescence label, a chemiluminescent label, a bioluminescent label, and enzymatic label, a chromogenic label and a radiolabel, and wherein detecting antibody -EphA2 binding comprises detecting at least one detectable label. claim 57 embodies the method of claim 47 wherein the cell population comprises cells selected from lung cells and colon cells. Claim 58 embodies the method of claim 47 wherein the cell population comprises epithelial cells. Claim 61 embodies the method of claim 61 wherein the cell population comprises metastatic cancer cells. Claim 64 embodies the method of claim 47 wherein the cell population comprises cells form a tissue biopsy. Claim 68 embodies the method of claim 47 wherein detecting reagent Eph-A2 binding comprises utilizing Western blot.

The abstract of Chen et al discloses that EphA2 is synonymous with Eck.

Easty et al teach a method for detecting metastatic melanoma cells in a cell population comprising the steps of lysing at least a portion of the cell population, incubating the lysed cells with a polyclonal antibody that specifically binds to Eck to allow antibody binding to Eck and detecting antibody-Eck binding by Western blot methodology using chemiluminescence as a detectable label (page 131, under the heading "Immunoblotting analysis"). Easty et al teach that an anti-Eck antibody was prepared by the method of Lindberg et al (Molecular and Cellular Biology, 1990, Vol. 10, pp. 6316-6324). Lindberg et al teach that antibodies were raised to the Eck protein by means of a fusion protein comprising residues 874 to 974 of Eck which are the 101 amino acids at the C-terminus (page 6321, first column, lines 4-7 under the heading "Eck has in vitro kinase activity and autophosphorylates on tyrosine residues") and is an intracellular

portion of Eck (figure 1 and page 6319, first column, lines 9-13 under the heading "eck encodes a new receptor PTK in the eph/elk family" and figure 3). Lindberg et al also teach that an anti-Eck antibody was made by binding to anti-Eck antibodies that bound to an Eck fusion protein as described by . Lindberg et al teach that Thus, it is inherent in the method of Easty et al that the anti-Eck antibody bound to an intracellular epitope of Eck. Easty et al also teach a method for detecting the presence of metastatic cells in a cell population comprising the steps of incubating the cells with a probe for Eck mRNA and detecting hybridization between the probe and the mRNA (page 130, under the heading "Northern blotting analysis"). Easty et al further teach that Eck is expressed at both the protein and the mRNA level in metastatic melanoma cell lines and in sections of metastatic melanomas as indicated by immunohistochemistry (page 135, second column lines 6-13), thus fulfilling the specific embodiments of claims 21 and 23 drawn to reagents which bind to nucleic acids embodiment the EphA2 protein and the specific embodiment of claim 52 drawn to a bound complex comprising a whole cell. Easty et al teach that elevated expression of Eck appeared to be correlated with metastasis to epithelial sites such as lung and ileum, rather than to non-epithelial sites such as lymph nodes and cutaneous deposits (page 132, second column, lines 14-19), thus fulfilling the specific embodiments of cell populations comprising lung, colon and epithelial cells.

Easty et al detect Eck protein by means of a polyclonal antiserum specific for Eck (page 131, first column, lines 10-12). The antiserum referenced by Easty et al was made by Lindberg et al against a fusion protein comprising amino acids 874-974 of Eck which comprises an intracellular domain of Eck. Easty et al do not teach the detection of metastatic melanoma cells using a monoclonal anti-Eck antibody.

Larrick et al teach the advantages of using a monoclonal antibody over a polyclonal antibody which include a constant propagating source (pages 7-8, under the heading IV).

It would have been prima facie obvious to one of skill in the art at the time the invention was made to make a monoclonal antibody that binds to amino acids 874-974 of Eck. One of skill in the art would have been motivated to do so by the teachings of Larrick et al on the convenience and predictability of monoclonal antibodies versus polyclonal serum.

Claims 1, 3, 5, 9-11, 33, 34, 35, 41, 43-45, 47, 49, 55, 56, 57, 58, 61, 64 and 66-68 are rejected under 35 U.S.C. 103(a) as being unpatentable over Easty et al (International Journal of Cancer, 1995, vol. 60, pp. 129-136) as evidenced by the abstract of Chen et al (Journal of Biological Chemistry, 1998, Vol. 273, pp. 24670-24675) and Lindberg et al (Molecular and Cellular Biology, 1990, vol. 10, pp. 6316-6324) in view of Larrick et al (In: Human Hybridomas and Monoclonal Antibodies, Engleman and Foung, Ed.s, 1985, pp. 8-9) as applied to claims 1, 3, 5, 11, 33, 34, 35, 41, 45, 47, 49, 55, 56, 57, 58, 61, 64 and 68 above, and further in view of the abstract of Muhlbauer et al (Clinical Cancer Research, May 1999, Vol. 5, pp. 1099-1105).

Claim 9 embodies the method of claim 1 wherein the cell population is harvested from a body fluid selected from the group consisting of blood, plasma, spinal fluid, saliva and urine. claim 10 embodies the method of claim 9 wherein the detecting step included a diagnostic method selected from the group consisting of ELISA and flow cytometry. Claim 43 embodies the method of claim 1 wherein the cell population comprises cells from a body fluid. Claim 44 embodies the method of claim 43 wherein the body fluid is selected from the group consisting of blood, plasma, spinal fluid, saliva and urine. Claim 66 embodies the method of claim 47 wherein the cell population comprises cells form a body fluid. Claim 67 embodies the method of claim 66 wherein the body fluid is selected from the group consisting of blood, plasma, spinal fluid, saliva and urine.

The combination of Easty et al and the abstract of Chen et al and Lindberg et al and Larrick et al render obvious the method of detecting metastatic melanoma by means of detecting the expression of EphA2 for the reasons set forth above. The combination of prior art references do not specifically teach the detection of metastatic melanoma cells in the blood by means of ELISA or flow cytometry.

The abstract of Muhlbauer et al teaches that metastatic melanoma cells can be detected in the blood of melanoma patients by means of an assay which includes ELISA.

It would have been prima facie obvious at the time the invention was made to use the monoclonal antibody which specifically binds to EPhA2 to detect metastatic melanoma cells in the peripheral blood of melanoma patients by means of the PCR-ELISA system. One of skill in the art would have been motivated to do so by the teachings of the abstract of Muhlbauer et al on

the sensitivity of the PCR-ELISA assay for the detection of the metastatic melanoma cells and the correlation between positive blood samples and tumor burden.

Claims 1, 3, 5, 11, 12, 33, 34, 35, 41, 45, 46, 47, 49, 55, 56, 57, 58, 61, 64 and 68 are rejected under 35 U.S.C. 103(a) as being unpatentable over Easty et al (International Journal of Cancer, 1995, vol. 60, pp. 129-136) as evidenced by the abstract of Chen et al (Journal of Biological Chemistry, 1998, Vol. 273, pp. 24670-24675) and Lindberg et al (Molecular and Cellular biology, 1990, vol. 10, pp. 6316-6324) in view of Larrick et al (In: Human Hybridomas and Monoclonal Antibodies, Engleman and Foung, Ed.s, 1985, pp. 8-9) as applied to claims 1, 3, 5, 11, 33, 34, 35, 41, 45, 47, 49, 55, 56, 57, 58, 61, 64 and 68 above and in further view of Easty et al (International Journal of Cancer, 1997, Vol. 71, pp. 1061-1065).

Claim 12 embodies the method of claim 11 further comprising the steps of providing a second antibody having phosphotyrosine specificity and Western blotting the second antibody. Claim 46 embodies the method of claim 1, wherein detecting the antibody-EphA2 binding comprises utilizing a Western blot; the method further comprising Western blotting with a second antibody having phosphotyrosine specificity.

Easty et al (1997) teaches that in addition to Eck, other protein tyrosine kinases are ectopically expressed in melanoma include Her2, Fgf-R4, Hek2, Tie, Tyro-9 and 10 and Axel (page 1061, first column, second full paragraph under the abstract, and title). Easty et al teach that the expression of the tyrosine kinases Ptk7 and Sek is lost in malignant melanoma. and that the expression of Kit and Tyro-3 is also decreased during melanoma progression but that Kdr and Met can be either increased or decreased in melanomas (page 1061, second column, lines 5-12).

It would have been prima facie obvious to one of skill in the art at the time the invention was made to use an antibody which would specifically bind another protein tyrosine kinase that was known to be ectopically expressed in malignant melanoma. or was known to have lost expression in malignant melanoma. One of skill in the art would have been motivated to do so by the teachings of Easty et al (1997) which indicate that the measurement of a single protein tyrosine kinase does not provide an absolute correlation with malignancy because while the trend is loss of tyrosine kinase expression, some tyrosine kinases are over expressed in some cell lines

and lost in other cells lines (page 1061, second column, lines 5-12). Thus, one of skill in the art would be moti9vated to measure the expression of more than one tyrosine kinase known to be ectopically expressed, lost or over expressed in malignant melanoma.

Claims 1, 3, 5, 6, 7, 11, 33, 34, 35, 41, 45, 47, 49, 55, 56, 57, 58, 61, 64 and 68 are rejected under 35 U.S.C. 103(a) as being unpatentable over Easty et al (International Journal of Cancer, 1995, vol. 60, pp. 129-136) and the abstract of Chen et al (Journal of Biological Chemistry, 1998, Vol. 273, pp. 24670-24675) and Lindberg et al (Molecular and Cellular biology, 1990, vol. 10, pp. 6316-6324) and Larrick et al (In: Human Hybridomas and Monoclonal Antibodies, Engleman and Foung, Ed.s, 1985, pp. 8-9) as applied to claims 1, 3, 5, 11, 33, 34, 35, 41, 45, 47, 49, 55, 56, 57, 58, 61, 64 and 68 above, and further in view of Kerr and Thorpe (LabFax Immunochemistry, 1994, pages 115 and 157).

Claim 6 embodies the method of claim 5 wherein the antibody is labeled with a fluorescent label and the detecting step comprises detecting a fluorescent label. Claim 7 embodies the method of claim 5 wherein the antibody is labeled with a radio active label and the detecting step comprising detecting a radioactive label.

Kerr and Thorpe summarizes routine methods for detecting antibodies bound to antigens which includes labeling of an antibody with a fluorescent label (page 157) or a radioactive label (page 115).

It would have been prima facie obvious to label the monoclonal anti-Eck antibody rendered obvious by the combination of Easty et al and the abstract of Chen and Lindberg et al and Larrick et al. One of skill in the art would have been motivated to do so by the teachings of Kerr and Thorpe on the routine methods available to label antibodies with fluorescent or radiolabels.

Claims 1, 3, 5, 11, 33, 34, 35, 41, 45, 47, 49, 51-53, 55, 56, 57, 58, 61, 64 and 68 are rejected under 35 U.S.C. 103(a) as being unpatentable over Easty et al (International Journal of Cancer, 1995, vol. 60, pp. 129-136) and the abstract of Chen et al (Journal of Biological Chemistry, 1998, Vol. 273, pp. 24670-24675) and Lindberg et al (Molecular and Cellular biology, 1990, vol. 10, pp. 6316-6324) and Larrick et al (In: Human Hybridomas and

Monoclonal Antibodies, Engleman and Foung, Ed.s, 1985, pp. 8-9) as applied to claims 1, 3, 5, 11, 33, 34, 35, 41, 45, 47, 49, 55, 56, 57, 58, 61, 64 and 68 above, and further in view of Kerr and Thorpe (Immunochemistry LabFax, 1994, pp. 191-197).

Claim 51 embodies the method of claim 47 wherein the antibody binds to an extracellular epitope of EphA2. Claim 52 embodies the method of claim 47 wherein the antbody-EphA2 complex is a complex comprising a whole cells. Claim 53 embodies the method of claim 52 wherein the detecting of the antibody-EphA2 binding comprises subjecting the bound complex to immunohistochemical staining.

Lindberg et al teach the amino acid sequence of the extracellular portion of Eck (Figure 1 and page 6319, first column, lines 9-13 under the heading "eck encodes a new receptor PTK in the eph/elk family") and the use of an anti-serum raised to the intracellular portion of eck in immunohistochemistry (page 6317, second column, sections "Antibodies" and "Immunohistochemistry"). Lindberg et al do not teach the use of a monoclonal antibody that binds an extracellular portion of Eckl in immunohistochemistry..

Kerr and Thorpe teach that immunofluorescent sating of surface antigens may be carried out on living cells with preservation of cell viability, and that because of the fluidity of membranes, intact antibodies can cross-link surface antigens resulting in the formation of patches which tend to form a collective 'cap'. Kerr and Thorpe teach that the capping phenomenon can be exploited to derive information on the association between molecules (page 197, under the heading "Surface labeling of living cells").

It would have been prima facie obvious at the time the claimed invention was made to use an antibody which binds to an extracellular eptiope of EphA2 for immunohistochemical staining of whole cells. One of skill in the art would have been motivated to do so by the teachings of Kerr and Thorpe regarding the binding of antibodies to surface antigens on living cells. One of skill in the art would know that binding of an antibody to an extracellular domain of EphA2 available on the surface of a cell within a tissue sample would be desirable in order to quickly visualize the presence of any metastatic cells expressing EphA2 and thereby avoiding procedures requiring fixing of the tissue in order to allow penetration of antibodies to bind to intracellular epitopes of EphA2 (page 191, section 3.1.2) or cell lysis to assay for epitopes of EphA2 on the intracellular domain.

Claims 8, 13, 36, 37, 42, 59, 60, 62, 63 and 65 are objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including all of the limitations of the base claim and any intervening claims.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Karen A Canella whose telephone number is (571)272-0828. The examiner can normally be reached on 10 a.m. to 9 p.m. M-F.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Yvonne Eyler can be reached on (571)272-0871. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Karen A. Canella, Ph.D.
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KAREN A. CANELLA PH.D
PRIMARY EXAMINER